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NEWS 16 AUG 28 ADISCTI Reloaded and Enhanced
NEWS 17 AUG 30 CA(SM)/CAPplus(SM) Austrian patent law changes
NEWS 18 SEP 11 CA/CAPplus enhanced with more pre-1907 records
NEWS 19 SEP 21 CA/CAPplus fields enhanced with simultaneous left and right
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=> s (muCANP? or muCL? or CANP? or CANPL? or CAPN? or calpain?)
L1 36634 (MUCANP? OR MUCL? OR CANP? OR CANPL? OR CAPN? OR CALPAIN?)

=> s (hcmv or cmv or cytomegalov?)
L2 146213 (HCMV OR CMV OR CYTOMEGALOV?)

=> s l1 and l2
L3 70 L1 AND L2

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 41 DUP REM L3 (29 DUPLICATES REMOVED)

=> s l4 and py<=2000
1 FILES SEARCHED...
L5 18 L4 AND PY<=2000

=> d l5 ibib abs 1-18

L5 ANSWER 1 OF 18 MEDLINE on STN
ACCESSION NUMBER: 2001023951 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10906334
TITLE: Antisense RNA-mediated deficiency of the calpain
protease, nCL-4, in NIH3T3 cells is associated with
neoplastic transformation and tumorigenesis.
AUTHOR: Liu K; Li L; Cohen S N
CORPORATE SOURCE: Department of Genetics and Department of Medicine, Stanford
University School of Medicine, Stanford, California
94305-5120, USA.
CONTRACT NUMBER: CA09302 (NCI)
HG 00044-04 (NHGRI)
SOURCE: The Journal of biological chemistry, (2000 Oct 6)
Vol. 275, No. 40, pp. 31093-8.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 22 Mar 2001
Last Updated on STN: 22 Mar 2001
Entered Medline: 13 Nov 2000

AB We previously have described the use of an antisense RNA strategy termed
random homozygous knock-out (RHKO) to identify negative regulators of cell
proliferation. Here we report the discovery that RHKO-mediated deficiency

of the nCL-4 **calpain** protease results in cellular transformation of and tumorigenesis by murine NIH3T3 fibroblasts. We isolated cell clones able to form colonies on 0.5% soft agar and found that these cells generated tumors when injected subcutaneously into nude mice. The gene inactivated by RHKO was identified as nCL-4 by genomic library screening, transcript analysis, and DNA sequencing. Anchorage-independent growth, as indicated by colony formation on soft agar, was reversed by reversal of antisense-mediated homozygous inactivation, but continued haplo-insufficiency of nCL-4 resulting from insertional mutagenesis of one nCL-4 allele was associated with persistent tumorigenesis. nCL-4 cDNA expressed in naive 3T3 cells in the antisense, but not sense, direction under control of the **cytomegalovirus** early promoter reproduced the anchorage-independent growth effects of RHKO. Our results implicate deficiency of the nCL-4 **calpain** protease in neoplastic transformation.

L5 ANSWER 2 OF 18 MEDLINE on STN
 ACCESSION NUMBER: 2000236682 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 10776924
 TITLE: Herpesviruses and periodontopathic bacteria in Trisomy 21 periodontitis.
 AUTHOR: Hanookai D; Nowzari H; Contreras A; Morrison J L; Slots J
 CORPORATE SOURCE: University of Southern California, School of Dentistry, Department of Periodontology, Los Angeles 90089-0641, USA.
 SOURCE: Journal of periodontology, (2000 Mar) Vol. 71, No. 3, pp. 376-84.
 Journal code: 8000345. ISSN: 0022-3492.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Dental Journals; Priority Journals
 ENTRY MONTH: 200006
 ENTRY DATE: Entered STN: 22 Jun 2000
 Last Updated on STN: 22 Jun 2000
 Entered Medline: 13 Jun 2000

AB BACKGROUND: Little is known about the etiology and pathogenesis of periodontal disease in Trisomy 21 patients. This study determined the occurrence of herpesviruses and putative periodontopathic bacteria in Trisomy 21 periodontitis. METHODS: Nineteen Trisomy 21 patients (17 to 37 years of age) contributed subgingival samples from molar and bicuspid teeth presenting interproximal periodontitis lesions (probing depths, 5 to 8 mm) and from shallow periodontal sites (probing depths, 1 to 3 mm). Samples were obtained at baseline, and at 1 and 4 weeks after subgingival debridement by means of hand instruments and ultrasonic scalers. Epstein-Barr virus type 1 and 2 (EBV-1 and EBV-2), human **cytomegalovirus** (HCMV), and herpes simplex virus (HSV) were identified by sensitive and specific nested polymerase chain reaction. Putative periodontopathic bacteria were identified by means of non-selective and selective culture. RESULTS: Of 19 Trisomy 21 periodontitis lesions, 6 (32%) were positive for EBV-1, 5 (26%) were positive for HCMV, 3 (16%) were positive for HSV, and 2 (11%) showed viral co-infection. Of 19 shallow periodontal sites, only one revealed HCMV. *Prevotella intermedia*, *Bacteroides forsythus*, and *Capnocytophaga* species were detected in higher proportions in deep than in shallow periodontal pockets ($P = 0.02$). Subgingival debridement did not reduce genomic herpesvirus presence but caused a decrease in proportions of *Porphyromonas gingivalis* and *Capnocytophaga* species. CONCLUSIONS: Periodontal herpesvirus-bacteria coinfections may play important roles in the pathogenesis of destructive periodontal disease in Trisomy 21 patients. Herpesviruses may reduce the periodontal defense and promote growth of subgingival bacteria capable of causing periodontal breakdown.

L5 ANSWER 3 OF 18 MEDLINE on STN

ACCESSION NUMBER: 1998344739 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9681414
TITLE: Atrial natriuretic peptide gene delivery attenuates hypertension, cardiac hypertrophy, and renal injury in salt-sensitive rats.
AUTHOR: Lin K F; Chao J; Chao L
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Medical University of South Carolina, Charleston 29425-2211, USA.
CONTRACT NUMBER: HL29397 (NHLBI)
HL56686 (NHLBI)
SOURCE: Human gene therapy, (1998 Jul 1) Vol. 9, No. 10, pp. 1429-38.
Journal code: 9008950. ISSN: 1043-0342.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199809
ENTRY DATE: Entered STN: 6 Oct 1998
Last Updated on STN: 14 Dec 2002
Entered Medline: 21 Sep 1998

AB To investigate potential therapeutic effects of atrial natriuretic peptide (ANP) gene delivery on renal and cardiac disorders, adenovirus harboring the human ANP gene (Ad.RSV-**cANP**) was delivered into Dahl salt-sensitive (DSS) rats on a high-salt diet. A single intravenous injection of the ANP gene caused a significant delay of blood pressure increase 3 days post-injection and the effect lasted for more than 5 weeks. A maximal blood pressure reduction of 32.8 mmHg was observed after ANP gene delivery, as compared with that of control rats injected with Ad.**CMV-LacZ**. Immunoreactive human ANP can be detected in the heart, lung, and kidney of rats after gene delivery. ANP gene delivery caused significant increases in renal blood flow, glomerular filtration rate, sodium output, urine excretion, and urinary cGMP levels. These beneficial effects were reflected morphologically by a reduction in cardiomyocyte size, attenuation of the glomerular-sclerotic lesions, tubular injury and arterial thickening. This study demonstrated the usefulness of somatic gene transfer as a new tool for ANP gene delivery in studying salt-related hypertension and renal and cardiovascular diseases. In addition, the findings also suggest that ANP gene delivery may have potential in therapeutic applications.

L5 ANSWER 4 OF 18 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:184941 BIOSIS
DOCUMENT NUMBER: PREV199900184941
TITLE: Human **cytomegalovirus**-activated **calpain** and p21Cip1 degradation in human lung fibroblasts.
AUTHOR(S): Chen, Z.; Knutson, E.; Kurosky, A.; Liu, S.; Albrecht, T.
CORPORATE SOURCE: Univ. Texas Med. Branch, Galveston, TX 77555, USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (March, 1999) Vol. 40, pp. 447-448. print.
Meeting Info.: 90th Annual Meeting of the American Association for Cancer Research. Philadelphia, Pennsylvania, USA. April 10-14, 1999. American Association for Cancer Research.
ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 5 May 1999
Last Updated on STN: 5 May 1999

L5 ANSWER 5 OF 18 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:272936 BIOSIS

DOCUMENT NUMBER: PREV199800272936
 TITLE: Infections occurring during the courses of anticancer chemotherapy in children with ALL: A retrospective analysis of 59 patients.
 AUTHOR(S): Rahiala, J. [Reprint author]; Perkkio, M.; Riikonen, P.
 CORPORATE SOURCE: Dep. Pediatr., Kuopio Univ. Hosp., FIN-70211 Kuopio, Finland
 SOURCE: Pediatric Hematology and Oncology, (March-April, 1998) Vol. 15, No. 2, pp. 165-174. print.
 CODEN: PHONEN. ISSN: 0888-0018.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 24 Jun 1998
 Last Updated on STN: 24 Jun 1998

AB In a retrospective analysis we evaluated the occurrence of infections in 59 children with acute lymphoblastic leukemia (ALL) during the entire duration of their anticancer chemotherapy. We recorded a total of 245 infection episodes, 118 (50%) being during neutropenia and 119 (50%) during nonneutropenia. The infections most commonly detected during neutropenia were fevers of undetermined origin (36%), clinically or microbiologically defined focal infections (33%), and bacteremias (28%). During nonneutropenia, upper respiratory tract infections (55%) were the most common. Patients needed hospitalization for infections for a total of 195 days (i.e., a mean of 33 days per patient) and the mean number of infection episodes was 4.2 per patient. Recurrent fever developed in 21% of the children with bacteremia. Mortality caused by bacteremias was 10%. Infections during the chemotherapy of ALL were a significant cause of morbidity in children, but mortality was low.

L5 ANSWER 6 OF 18 CA COPYRIGHT 2006 ACS on STN
 ACCESSION NUMBER: 144:32200 CA
 TITLE: Ricin-like toxin precursors cleavable by disease-specific proteinases for treatment of cancer, viral or parasitic infections
 INVENTOR(S): Borgford, Thor; Braun, Curtis; Purac, Admir; Stoll, Dominik
 PATENT ASSIGNEE(S): Can.
 SOURCE: U.S. Pat. Appl. Publ., 495 pp., Cont.-in-part of U.S. 89,058.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 4
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005272048	A1	20051208	US 2004-893584	20040719
WO 9849311	A2	19981105	WO 1998-CA394	19980430 <--
WO 9849311	A3	19990211		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6593132	B1	20030715	US 1999-403752	19991029
US 6803358	B1	20041012	US 2000-551151	20000414
WO 2001025267	A2	20010412	WO 2000-CA1162	20001004
WO 2001025267	A3	20020328		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,				

HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
 LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
 SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
 YU, ZA, ZW
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
 DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,
 CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 7060789 B1 20060613 US 2002-89058 20020919
 PRIORITY APPLN. INFO.: US 1997-45148P P 19970430
 US 1997-63715P P 19971029
 WO 1998-CA394 W 19980430
 US 1999-157807P P 19991004
 US 1999-403752 A2 19991029
 US 2000-197409P P 20000414
 US 2000-551151 A2 20000414
 WO 2000-CA1162 W 20001004
 US 2002-89058 A2 20020919

AB Ricin precursors with the ricin A and B chains linked by a protease-labile linker peptide are described for use in the treatment of disease. The linker peptide contains a cleavage site for a disease specific protease such as a cancer, viral or parasitic protease. The ricin A or B chains may be replaced by comparable cytotoxic proteins such as the abrin A chain. The protein is delivered to the target tissue using viral vectors carrying an expression cassette for the ricin fusion protein gene. Construction of a series of variants of preproricin cleavable by a number of different proteinases and their recombinant expression in yeast is described.

L5 ANSWER 7 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 141:325694 CA

TITLE: Ricin-like toxin variants comprising A chain and B chain linked with a linker for treatment of cancer, viral or parasitic infections

INVENTOR(S): Borgford, Thor

PATENT ASSIGNEE(S): Twinstrand Therapeutics Inc., Can.

SOURCE: U.S., 280 pp., Cont.-in-part of U.S. 6,593,132.

CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 4

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6803358	B1	20041012	US 2000-551151	20000414
WO 9849311	A2	19981105	WO 1998-CA394	19980430 <--
WO 9849311	A3	19990211		

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

US 6593132 B1 20030715 US 1999-403752 19991029

US 2005272048 A1 20051208 US 2004-893584 20040719

PRIORITY APPLN. INFO.: WO 1998-CA394 W 19980430
 US 1999-403752 A2 19991029
 US 1997-45148P P 19970430
 US 1997-63715P P 19971029
 US 1999-157807P P 19991004
 US 2000-197409P P 20000414
 US 2000-551151 A2 20000414

WO 2000-CA1162

W 20001004

US 2002-89058

A2 20020919

AB The present invention provides a protein having an A chain of a ricin-like toxin, a B chain of a ricin-like toxin and a heterologous linker amino acid sequence, linking the A and B chains. The linker sequence contains a cleavage recognition site for a disease specific protease such as a cancer, fungal, viral or parasitic protease. The invention also relates to a nucleic acid mol. encoding the protein and to expression vectors incorporating the nucleic acid mol. Also provided is a method of inhibiting or destroying mammalian cancer cells, cells infected with a virus, a fungus, or parasites, or parasites utilizing the nucleic acid mols. and proteins of the invention and pharmaceutical compns. for treating human cancer, viral infection, fugal infection, or parasitic infection.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 134:220676 CA

TITLE: Transcriptome analysis of fibroblast cells
immediate-early after human **cytomegalovirus**
infection

AUTHOR(S): Kenzelmann, Marc; Muhlemann, Kathrin

CORPORATE SOURCE: Institute of Medical Microbiology, University of Bern,
Bern, 3010, Switz.

SOURCE: Journal of Molecular Biology (2000), 304(5),
741-751

CODEN: JMOBAK; ISSN: 0022-2836

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Human **cytomegalovirus** (HCMV) has been shown to have the potential to alter cellular gene expression early after infection. However, one-gene approaches and the use of closed system gene expression technologies have identified only few cellular genes whose activity changed immediate-early. Therefore, serial anal. of gene expression (SAGE) was used to investigate the transcriptional program of human fibroblasts in response to HCMV in the immediate-early phase of infection. Differential expression of various cellular genes was monitored. Transcriptional expression changes of genes coding for ribosomal proteins reflected a general cellular response to starvation and stress. But differential regulation of genes coding for transcription factors and proteins associated with cellular metabolism, homeostasis and cell structure may represent transcriptional alterations in response to HCMV infection. Expression kinetics by 5' nuclease fluorogenic real-time PCR of selected genes revealed partial protection of infected cells against initial stress-associated alterations of gene expression and indicated fluctuations of transcriptional levels over time. Addnl., agreement with the quant. results obtained by SAGE was observed only for genes up-regulated in HCMV-infected cells. This finding pointed to various tech. and statistical parameters that all may be critical for quant. transcriptome studies using global approaches, especially when exploring biol. systems in a critical phase of cellular physiol. (c) 2000 Academic Press.

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 133:114745 CA

TITLE: **Calpain** inhibitor 1 activates p53-dependent
apoptosis in tumor cell lines

AUTHOR(S): Atencio, Isabella A.; Ramachandra, Murali; Shabram,
Paul; Demers, G. William

CORPORATE SOURCE: Canji, Inc., San Diego, CA, 92121, USA

SOURCE: Cell Growth & Differentiation (2000), 11(5),
247-253
CODEN: CGDIE7; ISSN: 1044-9523
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Reports suggest a role of **calpains** in degradation of wild-type p53, which may regulate p53 induction of apoptosis. A **calpain** inhibitor, n-acetyl-leu-leu-norleucinal (**calpain** inhibitor 1), was assessed for ability to enhance p53-dependent apoptosis in human tumor cell lines with endogenous wild-type p53 and in altered p53 cell lines with the replacement of wild-type p53 by a recombinant adenovirus (rAd-p53). **Calpain** inhibitor 1 treatment resulted in increased levels of activated p53, increased p21 protein, and activation of caspases. Cell lines with wild-type, but not mutated or null, p53 status arrested in G0/G1 and were sensitive to **calpain** inhibitor-induced apoptosis. Regardless of endogenous p53 status, **calpain** inhibitor treatment combined with rAd-p53, but not empty vector virus, enhanced apoptosis in tumor cell lines. These results demonstrate p53-dependent apoptosis induced by a **calpain** inhibitor and further suggest a role for **calpains** in the regulation of p53 activity and induction of apoptotic pathways.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 10 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 133:70691 CA

TITLE: Identifying protease modulators with α -donor fusion fusion proteins releasing α -galactosidase in results to cleavage activity

INVENTOR(S): Menzel, Rolf; Wang, Shaojie

PATENT ASSIGNEE(S): Small Molecule Therapeutics, Inc., USA

SOURCE: PCT Int. Appl., 59 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000039348	A1	20000706	WO 1999-US31026	19991223 <--
W:	AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
EP 1141419	A1	20011010	EP 1999-966678	19991223
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO			

PRIORITY APPLN. INFO.: US 1998-113589P P 19981224
WO 1999-US31026 W 19991223

AB The present invention relates to protease assays. More particularly, this invention relates to compds. and methods useful for assaying for protease activity. The invention relates to targeted, efficient and high-throughput screens to identify small mols. compds., peptides, etc. that modulate, i.e., interfere with or enhance, protease activity. The invention encompasses a variety of in vivo and in vitro assays. The assays comprise exposing an α -donor fusion polypeptide to a protease, wither within a cell or in a cell-free system, wherein the

α -donor polypeptide comprises an α -donor in operative assocn with a protease substrate, for a time sufficient to allow protease cleavage and release of β -galactosidase. Thus, the UL80 α -wt fusion proteins was constructed, comprising the first 11 amino acids from the plasmid vector, 708 amino acids of the UL80 polypeptide (the protein precursor domain) of human **cytomegalovirus**, a 7-amino acid linker, and 80 amino acids residues from the plasmid pUC19, of which the first 51 amino acids represent residues 4-55 of β -galactosidase (α -donor domain). A hepatitis C viral polyprotein α -fragment (NSFA protein) fusion protein is also constructed. The invention further encompasses therapeutic compds., such as antivirals, identified using the screening methods.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 132:344118 CA

TITLE: Adenoviral vectors with E1B deletion replicated in tumor cells and their use in cancer therapy

INVENTOR(S): Howe, John A.; Perry, Stuart T.

PATENT ASSIGNEE(S): Canji, Inc., USA

SOURCE: PCT Int. Appl., 46 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000029573	A2	20000525	WO 1999-US26003	19991117 <--
WO 2000029573	A3	20001005		
W:	AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MX, NO, NZ, PL, PT, RO, RU, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			

PRIORITY APPLN. INFO.: US 1998-195748 A 19981118

AB The present invention provides a replication competent recombinant adenovirus containing a constitutive viral or cellular promoter operably linked to a p53 gene, wherein said vector is defective in E1B55K function. The vectors of the present invention are capable of replication and lysis of neoplastic cells. The vectors may optionally include modifications to the genome so as to impart addnl. therapeutic or targeting functions. The present invention also provides pharmaceutical formulations of such vectors. The present invention further provides methods of use and preparing of such vectors.

L5 ANSWER 12 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 132:298826 CA

TITLE: **Calpain** inhibitors and p53 viral vectors to enhance apoptosis

INVENTOR(S): Atencio, Isabella A.; Laface, Drake M.; Ramachandra, Muralidhara; Shabram, Paul W.

PATENT ASSIGNEE(S): Canji, Inc., USA

SOURCE: PCT Int. Appl., 63 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000021575	A2	20000420	WO 1999-US21453	19991014 <--
WO 2000021575	A3	20001123		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MX, NO, NZ, PL, PT, RO, RU, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1998-172685 A 19981015

AB The present invention provides a method to enhance apoptosis in a cell by the administration of p53 in combination with a **calpain** inhibitor. The present invention provides a method of increasing the infectivity of a cell to a viral vector by treatment of the cell with a **calpain** inhibitor. The present invention further provides a method of enhancing transcription of a therapeutic transgene from the CMV promoter. The present invention also provides a method of suppressing the in vivo CTL response to viral vectors by the use of **calpain** inhibitors. The present invention further provides a pharmaceutical formulation of p53 and a **calpain** inhibitor in a pharmaceutically acceptable carrier. The present invention provides a method of ablating neoplastic cells in a mammalian organism in vivo by the co-administration of a **calpain** inhibitor and p53. The present invention also provides a method of ablating neoplastic cells in a population of normal cells contaminated by said neoplastic cells ex vivo by the administration of a recombinant adenovirus in combination with a **calpain** inhibitor to said population.

L5 ANSWER 13 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 132:133197 CA

TITLE: Novel methods for in vivo identification of enzyme inhibitors from random peptide-chymotrypsin inhibitor 2A (CI-2A) fusion library and their use in drug screening

INVENTOR(S): Halkier, Torben; Jespersen, Lene; Jensen, Allan

PATENT ASSIGNEE(S): M & E Biotech A/S, Den.

SOURCE: PCT Int. Appl., 136 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000005406	A1	20000203	WO 1999-DK408	19990716 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2335343	AA	20000203	CA 1999-2335343	19990716 <--
AU 9958985	A1	20000214	AU 1999-48985	19990716 <--
AU 751055	B2	20020808		
EP 1098991	A1	20010516	EP 1999-932689	19990716
EP 1098991	B1	20020911		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

TR 200100206	T2	20010621	TR 2001-200100206	19990716
EE 200100040	A	20020617	EE 2001-40	19990716
JP 2002521652	T2	20020716	JP 2000-561352	19990716
AT 223971	E	20020915	AT 1999-932689	19990716
NZ 509013	A	20021220	NZ 1999-509013	19990716
EP 1270746	A1	20030102	EP 2002-76171	19990716

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL

ZA 2001000195	A	20020108	ZA 2001-195	20010108
NO 2001000300	A	20010319	NO 2001-300	20010118

PRIORITY APPLN. INFO.:

DK 1998-956	A	19980720
US 1998-94868P	P	19980729
EP 1999-932689	A3	19990716
WO 1999-DK408	W	19990716

AB Novel methods (so called CellScreen® technol.) for in vivo identification enzyme inhibitors from random peptide-chymotrypsin inhibitor 2A (CI-2A) fusion library and their use in drug screening are described. Barley CI-2A from the potato inhibitor I family of protease inhibitors is used as the scaffold to display random peptide sequences in vivo since it can be stably and sufficiently expressed in the nucleus or ER of cultured cells, or displayed on the phage particles and remains biol. active. Random peptide library is constructed by inserting the random synthetic oligonucleotides or PCR fragments inside the CI-2A loop coding region in the retroviral expression vector and expressed intracellularly. The signal peptide sequence for various intracellular compartments or peptide tag can be fused at the N-terminus of the peptide-CI-2A library for the localization or purification purpose. The enzyme inhibitors or their relative RNA can be isolated from the phenotypically altered cells and used for further screening of their interaction partners which has therapeutic potentials.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 14 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 131:348774 CA

TITLE: Tandem fluorescent protein constructs and their preparation for enzyme assays

INVENTOR(S): Tsien, Roger Y.; Heim, Roger; Cubitt, Andrew

PATENT ASSIGNEE(S): The Regents of the University of California, USA; Aurora Biosciences Corporation

SOURCE: U.S., 33 pp., Cont.-in-part of U.S. Ser. No. 594,575.

CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 5

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5981200	A	19991109	US 1997-792553	19970131 <--
US 6803188	B1	20041012	US 1996-594575	19960131
PT 877805	T	20021031	PT 1997-905667	19970131
ES 2177939	T3	20021216	ES 1997-905667	19970131
US 2003186229	A1	20031002	US 2001-865291	20010524
US 6900304	B2	20050531		
US 2002164674	A1	20021107	US 2002-57505	20020125
US 2005026234	A1	20050203	US 2004-857622	20040528
PRIORITY APPLN. INFO.:			US 1996-594575	A2 19960131
			US 1997-792553	A1 19970131
			US 1999-396003	B2 19990913
			US 2001-865291	A2 20010524

AB This invention provides tandem fluorescent protein construct including a donor fluorescent protein moiety, an acceptor fluorescent protein moiety and a linker moiety that couples the donor and acceptor moieties. The

donor and acceptor moieties exhibit fluorescence resonance energy transfer which is eliminated upon cleavage. The constructs are useful in enzymic assays. Mutant green fluorescent proteins (GFPs) were created by mutagenesis of the Aequorea victoria GFP. Polyhistidine tagged tandem green and blue fluorescent proteins were recombinantly constructed having an inserted peptide sequence including cleavage recognition sites for many proteases. Cleavage expts. were done with trypsin, enterokinase and calpain.

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 15 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 131:333006 CA

TITLE: Production of recombinant replication-deficient viral vectors encoding exogenous transgenes via microcarrier-based process

INVENTOR(S): Giroux, Daniel D.; Goudreau, Ann M.; Ramachandra, Muralidhara; Shabram, Paul W.

PATENT ASSIGNEE(S): Canji, Inc., USA

SOURCE: PCT Int. Appl., 32 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9957297	A1	19991111	WO 1999-US9813	19990504 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MD, MG, MK, MN, MX, NO, NZ, PL, PT, RO, RU, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UZ, VN, YU, ZA				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 5994134	A	19991130	US 1998-73076	19980504 <--
CA 2328084	AA	19991111	CA 1999-2328084	19990504 <--
AU 9938823	A1	19991123	AU 1999-38823	19990504 <--
EP 1078095	A1	20010228	EP 1999-921681	19990504
EP 1078095	B1	20060308		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, LT, LV, FI, RO				
JP 2002513583	T2	20020514	JP 2000-547250	19990504
AT 319844	E	20060315	AT 1999-921681	19990504
EP 1657309	A1	20060517	EP 2006-2556	19990504
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, LT, LV, FI, RO, CY				
ES 2257861	T3	20060801	ES 1999-921681	19990504
PRIORITY APPLN. INFO.:				
			US 1998-73076	A 19980504
			EP 1999-921681	A3 19990504
			WO 1999-US9813	W 19990504

AB The present invention is directed to a method of producing recombinant viral vectors at high titers incorporating a variety of important advancements over the art. The method of the present invention incorporates multiple features which provide enhanced production of viruses, particularly those viruses encoding exogenous transgenes. The specifically illustrated method describes a method for the high titer serum-free media production of recombinant replication defective adenoviruses containing an exogenous transgene. The invention provides methods of preparing microcarriers, methods for seeding bioreactors at high cell d., increasing the infectivity of the producer cells to the virus, methods to increase product yield through synchronization of the cell cycle of the producer cells, and methods to minimize the deleterious effects of exogenous

transgenes. The invention further provides producer cells prepared by the process of the invention. The invention further provides viruses produced by the process.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 16 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 130:308784 CA

TITLE: Novel fluorescent reporter molecules and their applications including assays for caspases

INVENTOR(S): Weber, Eckard; Cai, Sui Xiong; Keana, John F. W.; Drewe, John A.; Zhang, Han-Zhong

PATENT ASSIGNEE(S): Cytovia, Inc., USA

SOURCE: PCT Int. Appl., 203 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9918856	A1	19990422	WO 1998-US21231	19981009 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2308125	AA	19990422	CA 1998-2308125	19981009 <--
AU 9910722	A1	19990503	AU 1999-10722	19981009 <--
AU 754634	B2	20021121		
EP 1026988	A1	20000816	EP 1998-953317	19981009 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2001519368	T2	20011023	JP 2000-515498	19981009
NZ 503619	A	20011130	NZ 1998-503619	19981009
US 6342611	B1	20020129	US 1998-168888	19981009
BR 9814816	A	20040622	BR 1998-14816	19981009
US 6335429	B1	20020101	US 2000-521650	20000308
US 2002150885	A1	20021017	US 2001-947387	20010907
US 6759207	B2	20040706		
US 2004191844	A1	20040930	US 2004-829381	20040422
PRIORITY APPLN. INFO.:				
			US 1997-61582P	P 19971010
			US 1998-33661	A 19980303
			US 1998-145746P	P 19980303
			US 1998-168888	A3 19981009
			WO 1998-US21231	W 19981009
			US 2001-947387	A3 20010907

OTHER SOURCE(S): MARPAT 130:308784

AB The present invention relates to novel fluorescent dyes, novel fluorogenic and fluorescent reporter mols. and new enzyme assay processes that can be used to detect the activity of caspases and other enzymes involved in apoptosis in whole cells, cell lines and tissue samples derived from any living organism or organ. The reporter mols. and assay processes can be used in drug screening procedures to identify compds. which act as inhibitors or inducers of the caspase cascade in whole cells or tissues. The reagents and assays described herein are also useful for determining the chemosensitivity of human cancer cells to treatment with chemotherapeutic drugs. The present invention also relates to novel fluorogenic and fluorescent reporter mols. and new enzyme assay processes that can be used to detect the activity of type 2 methionine aminopeptidase, dipeptidyl

peptidase IV, **calpain**, aminopeptidase, HIV protease, adenovirus protease, HSV-1 protease, **HCMV** protease and HCV protease. Caspase-3 substrate, N-Ac-DEVD-N'-octyloxycarbonyl Rhodamine 110 (preparation given), was used to stain apoptotic HL-60 cells.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 17 OF 18 CA COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 130:10614 CA

TITLE: Ricin precursors cleavable by disease-specific proteinases for treatment of cancer, viral or parasitic infections

INVENTOR(S): Borgford, Thor

PATENT ASSIGNEE(S): De Novo Enzyme Corp., Can.

SOURCE: PCT Int. Appl., 352 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 4

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9849311	A2	19981105	WO 1998-CA394	19980430 <--
WO 9849311	A3	19990211		
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG			
CA 2288943	AA	19981105	CA 1998-2288943	19980430 <--
AU 9870237	A1	19981124	AU 1998-70237	19980430 <--
EP 977862	A2	20000209	EP 1998-916743	19980430 <--
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI			
JP 2001523961	T2	20011127	JP 1998-546437	19980430
US 6593132	B1	20030715	US 1999-403752	19991029
US 6803358	B1	20041012	US 2000-551151	20000414
US 2004009551	A1	20040115	US 2003-394511	20030324
US 2005272048	A1	20051208	US 2004-893584	20040719

PRIORITY APPLN. INFO.:

US 1997-45148P	P	19970430
US 1997-63715P	P	19971029
WO 1998-CA394	W	19980430
US 1999-157807P	P	19991004
US 1999-403752	A2	19991029
US 2000-197409P	P	20000414
US 2000-551151	A2	20000414
WO 2000-CA1162	W	20001004
US 2002-89058	A2	20020919

AB Ricin precursors with the ricin A and B chains linked by a protease-labile linker peptide are described for use in the treatment of disease. The linker peptide contains a cleavage site for a disease specific protease such as a cancer, fungal, viral or parasitic protease. The ricin A chain may be replaced by comparable cytotoxic proteins such as the abrin A chain. The protein is delivered to the target tissue using viral vectors carrying an expression cassette for the ricin fusion protein gene. Construction of a series of variants of preproricin cleavable by a number of different proteinases is described. Cleavage and activation of these variants with the expected patterns of cleavage of rRNA is demonstrated.

L5 ANSWER 18 OF 18 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights

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ACCESSION NUMBER: 86129624 EMBASE
DOCUMENT NUMBER: 1986129624
TITLE: Overview of the problem of infections in the
immunocompromised host.
AUTHOR: Bodey G.P.
CORPORATE SOURCE: Department of Internal Medicine, The University of Texas
system Cancer Center, M.D. Anderson Hospital, Houston, TX
77030, United States
SOURCE: American Journal of Medicine, (1985) Vol. 79, No. 5 B, pp.
56-61. .
CODEN: AJMEAZ
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 / Drug Literature Index
006 Internal Medicine
004 Microbiology
030 Pharmacology
026 Immunology, Serology and Transplantation
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER